

NORTH DAKOTA DEPARTMENT of **HEALTH**









Tonight's Agenda

6:00 – 7:00	Hypertension Update	Dr. Vincent Canzanello, M.D.
7:00 – 8:00	Stress Management Training and Cardiovascular Health: Have We Found the Holy Grail?	Dr. Jon Ulven, Ph.D., L.P.

2nd Annual Hypertension Summit March 16th, 2017 Fargo, North Dakota

Hypertension Update

Vincent J. Canzanello, M.D.

Consultant, Division of Nephrology and Hypertension

Professor or Medicine

College of Medicine, Mayo Clinic



DISCLOSURES

Relevant Financial Relationship(s)

None

Off Label Usage
None



A professor is one who talks in someone else's sleep. *Anonymous*

Topics

- Systolic Blood Pressure Intervention Trial (SPRINT): a clinical practice changing study?
- Heart Outcomes Prevention Evaluate 3 (HOPE 3): Can statins and/or antihypertensive drugs lower CV risk in a population without dyslipidemia or hypertension and without baseline CVD?
- What is the best add-on drug in patients with resistant hypertension?
- Allopurinol: new uses for an old drug?



In an otherwise healthy 80 year old patient with hypertension, the Systolic Blood Pressure Intervention Trial (SPRINT) demonstrated that reducing the SBP to 120/80 resulted in all but one of the following:

- A. A significant reduction in cardiovascular and all cause mortality
- B. This benefit occurred regardless of baseline frailty status and/or gate speed
- C. A higher incidence of acute kidney injury but not in eventual renal outcomes (50% decrease in eGFR, dialysis, or transplant)
- D. More syncope and orthostatic hypotension
- **E.** More injurious falls



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- E. More injurious falls



The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812

NOVEMBER 26, 2015

VOL. 373 NO. 22

A Randomized Trial of Intensive versus Standard Blood-Pressure Control

The SPRINT Research Group*

Systolic Blood Pressure Intervention Trial (SPRINT) Research Group

Wright JT et al. NEJM 2015;373:2103-16.



Systolic Blood Pressure Intervention Trial (SPRINT)

- Randomized 9361 persons ≥ 50 y/o with SBP ≥130 mm Hg (most on medications) and increased CV risk* but no DM, to SBP target <120 mm Hg (intensive) or <140 mm Hg (standard treatment)
- Primary composite outcome MI, other ACS, stroke, heart failure, death from CV cause

*One or more of the following: clinical or subclinical CVD other than stroke; CKD excluding PCKD with an eGFR 20 to <60 ml/min/1.73 m² (4v MDRD), a 10-year risk of CVD of ≥15% by Framingham risk score; or age ≥75 years. Patients with DM or prior stroke excluded.



Systolic Blood Pressure Intervention Trial (SPRINT)

Good separation in achieved systolic BP

Mean numbers of medications were 2.8 in intensive and 1.8 in standard treatment groups

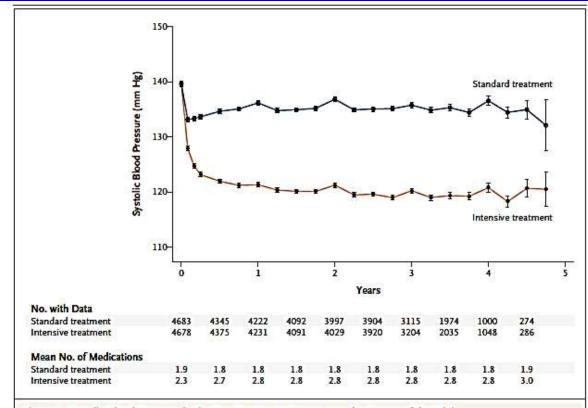


Figure 2. Systolic Blood Pressure in the Two Treatment Groups over the Course of the Trial.

The systolic blood-pressure target in the intensive-treatment group was less than 120 mm Hg, and the target in the standard-treatment group was less than 140 mm Hg. The mean number of medications is the number of blood-pressure medications administered at the exit of each visit. I bars represent 95% confidence intervals.

Systolic Blood Pressure Intervention Trial (SPRINT)

- Stopped early due to significantly lower rate of primary composite outcome of 1.65% per year in the intensive-treatment and 2.19% per year in standardtreatment group (HR with intensive treatment, 0.75; 95% confidence interval [CI], 0.64 to 0.89; P<0.001)
- All-cause mortality significantly lower in the intensive-treatment group (HR 0.73; 95% CI, 0.60 to 0.90; P = 0.003)

Wright JT et al. NEJM 2015;373:2103-16.



TABLE 1
Antihypertensive medications used in SPRINT

Medication class	Intensive therapy (%)	Standard therapy (%)	
Angiotensin-converting enzyme inhibitors or angiotensin II receptor blockers	76.7	55.2	
Thiazide-type diuretics	54.9	33.3	
Dihydropyridine calcium channel blockers	52.8	31.3	
Beta-blockers	41.1	30.8	
Aldosterone antagonists	8.7	4.0	
Other potassium-sparing diuretics	3.1	2.5	
Nondihydropyridine calcium channel blockers	4.7	4.3	
Direct vasodilators	7.3	2.4	

Information from SPRINT Research Group; Wright JT Jr, Williamson JD, Whelton PK, et al.
A randomized trial of intensive versus standard blood-pressure control.
N Engl J Med 2015; 373:2103–2116.



SPRINT results and adverse events

TABLE 2 SPRINT results at a glar	nce		
		per year	
Outcome	Intensive therapy	Standard therapy	Hazard ratio
Primary outcome ^a	1.65	2.19	0.75b
Secondary outcomes			
Myocardial infarction	0.65	0.78	0.83
Other acute coronary syndromes	0.27	0.27	1.00
Stroke	0.41	0.47	0.89
Heart failure	0.41	0.67	0.62 ^b
Cardiovascular mortality	0.25	0.43	0.57⁵
Other secondary outcomes			
All-cause mortality	1.03	1.40	0.73 ^b
In patients with chronic kidney disease—decrease in eGFR of ≥ 50% or end-stage renal disease	0.33	0.36	0.89
In patients without chronic kidney disease—decrease in eGFR of ≥ 30% to < 60 mL/min/1.73 m ²	1.21	0.35	3.49b

	Percent o			
Adverse events	Intensive therapy	Standard therapy	Hazard ratio	
Hypotension	3.4	2.0	1.70b	
Syncope	3.5	2.4	1.44b	
Hyponatremia	3.8	2.1	1.76b	
Hypokalemia	2.4	1.6	1.50b	
Injurious fall	7.1	7.1	1.00	
Orthostatic hypotension without dizziness	16.6	18.3	0.88°	
Orthostatic hypotension with dizziness	1,3	1.5	0.85	
Acute kidney injury	4.4	2.6	1.71 ^b	

*The composite of myocardial infarction, acute coronary syndrome, stroke, heart failure, or death from cardiovascular causes.
*P < .05.</p>

eGFR = estimated glomerular filtration rate, according to the Modification of Diet In Renal Disease study equation.

Information from SPRINT Research Group; Wright JT Jr, Williamson JD, Whelton PK, et al.

A randomized trial of intensive versus standard blood-pressure control.

N Engl J Med 2015; 373:2103–2116.



Is it really a good idea to lower BP to 120/80 mm Hg in patients 75 years and older?





Original Investigation | April 2014

Antihypertensive Medications and Serious Fall Injuries in a Nationally Representative Sample of Older Adults Mary E. Tinetti, MD1,2; Ling Han, MD, PhD1; David S. H. Lee, PharmD, PhD3; Gail J. McAvay, PhD1; Peter Peduzzi, PhD2; Cary P. Gross, MD1; Bingqing Zhou, PhD2; Haiqun Lin, PhD2

[+] Author Affiliations

JAMA Intern Med. 2014;174(4):588-595. doi:10.1001/jamainternmed.2013.14764.

Original Investigation

Intensive vs Standard Blood Pressure Control and Cardiovascular Disease Outcomes in Adults Aged ≥75 Years A Randomized Clinical Trial

Jeff D. Williamson, MD, MHS; Mark A. Supiano, MD; William B. Applegate, MD, MPH; Dan R. Berlowitz, MD; Ruth C. Campbell, MD, MSPH; Glenn M. Chertow, MD; Larry J. Fine, MD; William E. Haley, MD; Amret T. Hawfield, MD; Joachim H. Ix, MD, MAS; Dalane W. Kitzman, MD; John B. Kostis, MD; Marie A. Krousel-Wood, MD; Lenore J. Launer, PhD; Suzanne Oparil, MD; Carlos J. Rodriguez, MD, MPH; Christianne L. Roumie, MD, MPH; Ronald I. Shorr, MD, MS; Kaycee M. Sink, MD, MAS; Virginia G. Wadley, PhD; Paul K. Whelton, MD; Jeffrey Whittle, MD; Nancy F. Woolard; Jackson T. Wright Jr, MD, PhD; Nicholas M. Pajewski, PhD; for the SPRINT Research Group

Williamson et al. JAMA 2016;315:2673-2682



Background

- In the US: 75% of persons over 75 years have hypertension
- Benefits of antihypertensive treatment are conflicting
 - Systolic hypertension in the elderly program (SHEP): benefits were limited to participants without a self-reported physical ability limitation
 - Hypertension in the very elderly trial (HYVET): consistent benefit regardless of frailty status



Baseline characteristics of patients 75 years or older

- Intensive treatment (SBP< 120), n=1317
- Standard treatment (SBP<140), n=1319

	IT	ST
Gait speed		
Median (IQR), m/s	0.90 (0.77-1.05)	0.92 (0.77-1.06)
Speed <0.8 m/s, No. (%)	371 (28.2)	369 (28.0)
Frailty index, median (IQR) ^c	0.18 (0.13-0.23)	0.17 (0.12-0.22)
Frailty status, No. (%)		
Fit (frailty index ≤0.10)	159 (12.1)	190 (14.4)
Less fit (frailty index >0.10 to ≤0.21)	711 (54.0)	745 (56.5)
Frail (frailty index >0.21)	440 (33.4)	375 (28.4)
Montreal Cognitive Assessment score, median (IQR) ^d	22.0 (19.0-25.0)	22.0 (19.0-25.0)



Outcomes

- BPs: intensive: 123/62 standard: 135/67
- Intensive: 1 additional drug, 121 vs. 124 mm Hg in fit vs. frail, no difference in standard group
- The absolute rate of serious adverse events was higher, but not statistically different in the intensive vs. the standard treatment group for
 - Hypotension (2.4 vs. 1.4%)
 - Syncope (3.0 vs. 2.4%)
 - Electrolyte abnormalities (4.0 vs. 2.7%)
 - Acute kidney injury (5.5 vs. 4.0%)
- The were no differences in the incidences of injurious falls or orthostatic hypotension



Outcomes (continued)

Table 3. Incidence of Cardiovascular, Renal, and Mortality Outcomes by Treatment Group								
	Intensive Treatment		Standard Treatment					
	No. With Outcome Events (n = 1317) ^a	% (95% CI) With Outcome Events/y	No. With Outcome Events (n = 1319) ^a	% (95% CI) With Outcome Events/y	HR (95% CI) ^b	P Value		
All participants								
Cardiovascular disease primary outcome ^c	102	2.59 (2.13-3.14)	148	3.85 (3.28-4.53)	0.66 (0.51-0.85)	.001		
Myocardial infarction (MI) ^d	37	0.92 (0.67-1.27)	53	1.34 (1.02-1.75)	0.69 (0.45-1.05)	.09		
ACS not resulting in MI ^d	17	0.42 (0.26-0.68)	17	0.42 (0.26-0.68)	1.03 (0.52-2.04)	.94		
Stroke ^d	27	0.67 (0.46-0.97)	34	0.85 (0.61-1.19)	0.72 (0.43-1.21)	.22		
Heart failure ^d	35	0.86 (0.62-1.20)	56	1.41 (1.09-1.83)	0.62 (0.40-0.95)	.03		
Cardiovascular disease death ^d	18	0.44 (0.28-0.70)	29	0.72 (0.50-1.03)	0.60 (0.33-1.09)	.09		
Nonfatal MI	37	0.92 (0.67-1.27)	53	1.34 (1.02-1.75)	0.69 (0.45-1.05)	.09		
Nonfatal stroke	25	0.62 (0.42-0.91)	33	0.83 (0.59-1.16)	0.68 (0.40-1.15)	.15		
Nonfatal heart failure	35	0.86 (0.62-1.20)	55	1.39 (1.06-1.81)	0.63 (0.40-0.96)	.03		
All-cause mortality	73	1.78 (1.41-2.24)	107	2.63 (2.17-3.18)	0.67 (0.49-0.91)	.009		
Primary outcome plus all-cause mortality	144	3.64 (3.09-4.29)	205	5.31 (4.63-6.09)	0.68 (0.54-0.84)	<.001		

Conclusions

- Lowering SBP to the 120s in the elderly reduces CV events and all cause mortality regardless of frailty status (despite a mean DBP of 62)
- Generally well tolerated
- How to approach management of hypertension in a community dwelling patient 75 years or older and without baseline orthostatic hypotension (SBP< 110 after 1 minute standing):
 - Begin with an initial SBP goal of <140 and if tolerated well, try for <130



In a 65 year old man without known cardiovascular disease, BP 138/82 and LDL-C 127, what may be an effective strategy to reduce cardiovascular risk?

- A. rosuvastatin 10 mg/d
- B. candesartan 16 mg and hctz 12.5 mg/d
- C. both
- D. neither



In a 65 year old man without known cardiovascular disease, BP 138/82 and LDL-C 127, what may be an effective strategy to reduce cardiovascular risk?

- A. rosuvastatin 10 mg/d
- B. candesartan 16 mg and hctz 12.5 mg/d
- C. both
- D. neither



The "polypill": a strategy to reduce cardiovascular disease by more than 80% Wald NJ, Law MR, BMJ 2003;326:1419

- Heart attack, stroke, other preventable cardiovascular diseases kill or seriously affect half the population in Western societies
- Drug treatment to prevent IHD and stoke have been limited to single risk factors, targeting the minority of patients in the tail end of risk factor distribution and reducing them to the level of the "average" population
- 96% of deaths from IHD and stroke occur in people ≥ 55 yrs with average or high normal risk factor values (average age is 60-65 yrs) therefore to meaningfully reduce CV disease morbidity and mortality, everyone 55 or older needs to be treated with drugs



The polypill

- Contents
 - Statin: 10 mg atorvastatin or other equivalent
 - 3 BP drugs (1/2 usual starting dose)
 - Hctz, atenolol, and an ACEI
 - Folic acid (remember this is 2003)
 - Aspirin (low dose)
- Risk reduction and adverse events (age ≥ 55 yrs) were based on results of individual clinical trials of each drug separately-and then multiplying the relative risks of each agent for a combined effect
- Years of life gained calculated from population studies/registry data of longevity in people 55 or older who did not have a vascular disease event



The polypill: risk reduction

Table 1 Effects of the Polypill on the risks of ischaemic heart disease (IHD) and stroke after two years of treatment at age 55-64

			% reduction in		
Risk factor	Agent	Reduction in risk factor	IHD event	Stroke	Source of evidence
LDL cholesterol	Statin†	1.8 mmol/l (70 mg/dl) reduction in LDL cholesterol	61 (51 to 71)	17 (9 to 25)	Law et al ¹
Blood pressure	Three classes of drug at half standard dose	11 mm Hg diastolic	46 (39 to 53)	63 (55 to 70)	Law et al ¹⁶
Serum homocysteine	Folic acid (0.8 mg/day)	3 μmol/l	16 (11 to 20)	24 (15 to 33)	Wald et al ⁹
Platelet function	Aspirin (75 mg/day)	Not quantified	32 (23 to 40)	16 (7 to 25)	Table A on bmj.com
Combined effect	All		88 (84 to 91)	80 (71 to 87)	

LDL=low density lipoprotein.

Wald NJ, Law MR, BMJ 2003;326:1419



^{*95%} confidence intervals include imprecision of the estimates of both the agent reducing the risk factor and the risk factor reduction decreasing risk. †Atorvastatin 10 mg/day, or simvastatin or lovastatin 40 mg/day taken in the evening or 80 mg/day taken in the morning.

The polypill: proportion of people who would benefit and years of event-free life gained

Table 2 Expected benefits in 100 men and 100 women without a known vascular disease who start taking the Polypill at age 55. Calculations are based on a Markov model and allow for other causes of death

	M	en	Women		
Age (years)	No who benefit*	Years gained†	No who benefit*	Years gained†	
Up to 65	7	21	3	24	
Up to 75	18	16	11	18	
Up to 85	30	13	24	14	
Up to any age	36	12	35	11	

^{*}Ischaemic heart disease event or stroke avoided or delayed.

Adverse events:

8% if 3 best tolerated bp drugs used: hctz, ARB, CCB15% if 3 previously described, least expensive bp drugs used



[†]Average years of life gained without an IHD event or stroke in those who benefit.

The polypill: conclusions (provocative to say the least)

- In people 55 or older: may prevent 88% or heart attacks, 80% of strokes and 1 in 3 would gain an average of 11-12 years without having a heart attack or stroke
- All people 55 or older should be treated.
- No need for baseline risk factor measurements (LDL-C, BP, homocysteine, platelet function) since intervention should be effective regardless of baseline values
- No need for monitoring in most patients in absence of symptoms or particular concern for events eg asthma-BB, renal disease-ACEI or ARB, etc
- Should be low-cost since all drugs now generic
- If a drug formulation prevented all cancers and was safe, would it be widely used?



The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Blood-Pressure and Cholesterol Lowering in Persons without Cardiovascular Disease

Salim Yusuf, M.B., B.S., D.Phil., Eva Lonn, M.D., Prem Pais, M.D.,
Jackie Bosch, Ph.D., Patricio López-Jaramillo, M.D., Ph.D., Jun Zhu, M.D.,
Denis Xavier, M.D., Alvaro Avezum, M.D., Ph.D., Lawrence A. Leiter, M.D.,
Leopoldo S. Piegas, M.D., Ph.D., Alexander Parkhomenko, M.D., Ph.D.,
Matyas Keltai, M.D., Ph.D., Katalin Keltai, M.D., Ph.D., Karen Sliwa, M.D., Ph.D.,
Irina Chazova, M.D., Ph.D., Ron J.G. Peters, M.D., Ph.D., Claes Held, M.D., Ph.D.,
Khalid Yusoff, M.D., Basil S. Lewis, M.D., Petr Jansky, M.D.,
Kamlesh Khunti, M.D., Ph.D., William D. Toff, M.D., Christopher M. Reid, Ph.D.,
John Varigos, B.Sc., Jose L. Accini, M.D., Robert McKelvie, M.D., Ph.D.,
Janice Pogue, Ph.D.,* Hyejung Jung, M.Sc., Lisheng Liu, M.D., Rafael Diaz, M.D.,
Antonio Dans, M.D., and Gilles Dagenais, M.D., for the HOPE-3 Investigators*

N Engl J Med 2016;374:2032-43



HOPE-3

- N=12,705 pts (men 55 or older, women 65 or older with at least one CV risk factor though women could be 60 or older with 2 risk factors):
 - Elevated waist-to-hip ratio
 - Low HDL-C
 - Impaired glucose tolerance
 - Current or recent tobacco use
 - FH premature CAD
 - Early renal dysfunction
- No indication or contraindication to study drugs
- Could have hypertension but not on an ACEI, ARB, or thiazide diuretic
- There were no specific lipid or BP levels mandated for entry



HOPE-3

- 228 centers, 21 countries
- Patients randomized to:
 - Candesartan 16 mg plus HCTZ 12.5 mg or placebo
 - Rosuvastatin 10 mg or placebo
- No specific LDL-C or BP targets
- Outcomes:
 - First coprimary: composite of death from CVD, nonfatal MI, nonfatal stroke
 - Second coprimary: composite of above plus resuscitated cardiac arrest, heart failure, or revascularization
 - Secondary outcome: second coprimary plus angina with evidence of ischemia
- Planned mean f/u of 5 years

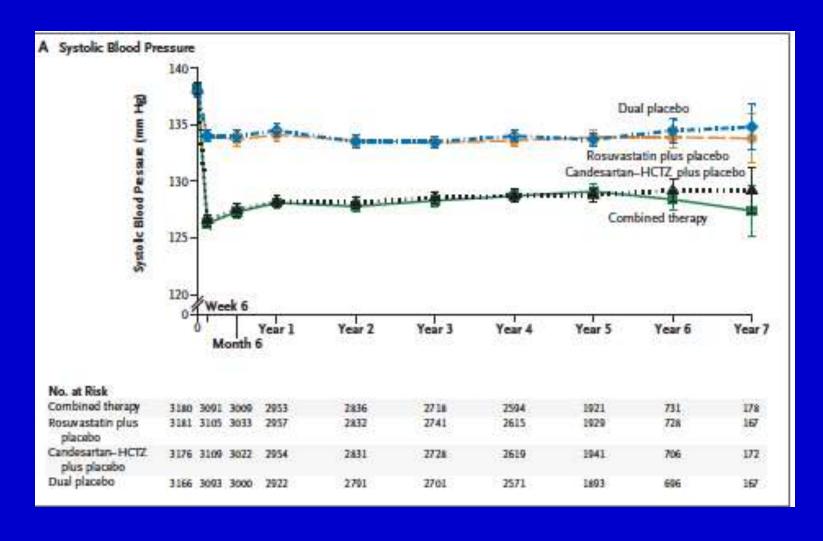


HOPE-3

- Baseline characteristics:
 - Mean age 65.7 yrs (46.2% women)
 - Mean SBP 138 mm Hg
 - Mean LDL-C 128 mg/dl
- Mean f/u 5.6 yrs
- Adherence: at end of trial, 75% in dual Rx group remained on Rx vs
 72% for the dual placebo group
- Adverse effects: muscle weakness and dizziness higher in the combined-therapy group



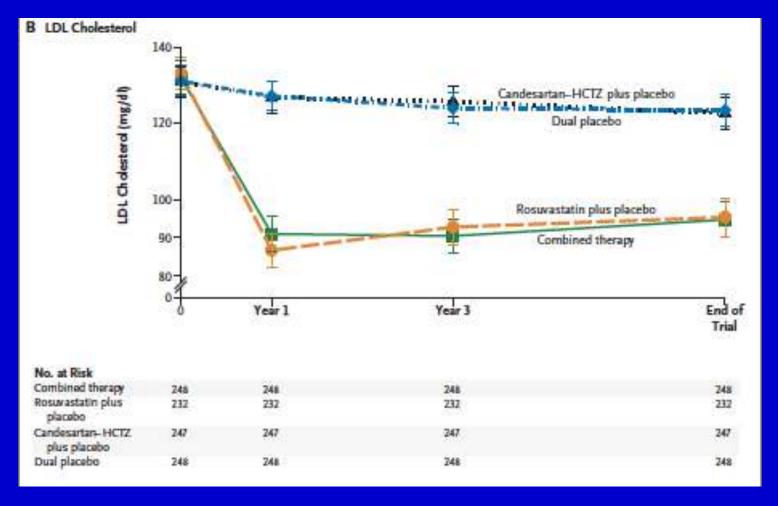
Systolic blood pressure



Mean decrease: 6.2 Hg mm, combined Rx vs dual placebo



LDL-cholesterol



Mean decrease: 33.7 mg/dL, combined Rx vs dual placebo



Coprimary and secondary outcomes

Table 2. Primary, Secondary, and Other Outcomes.*						
Outcome	Candesartan- Hydrochlorothiazide plus Rosuvastatin (N = 3180)	Ros uvastatin plus Placebo (N = 3181)	Candesartan- Hydrochlorothiazide plus Placebo (N=3176)	Placebo plus Placebo (N = 31 68)	Candesartan-Hydrochloro Rosuvastatin v s. Placebo	
					Hazard Ratio (95% CI)	P Value
Coprimary outcomes — no. (%)	PESTONAN	NW STOWN	No-MARKA	Wo-StaWA	2010 CONT CONT CONT CONT CONT CONT CONT CONT	25.52
First coprimary outcome	113 (3.6)	122 (3.8)†	147 (4.6) ‡	157 (5.0)	0.71 (0.56-0.90)	0.005
Second coprimary outcome	136 (4.3)	141 (4.4)	176 (5.5)¶	187 (5.9)	0.72 (0.57-0.89)	0.003
Secondary outcome — no. (%)	147 (4.6)	159 (5.0)	188 (5.9)	205 (6.5)	0.71 (0.57-0.87)	0.001
Components of the coprimary and secondary outcomes — no	. (%)			222112000		
Death from cardiov ascular causes	75 (24)	79 (2.5)	80 (2.5)	91 (2.9)	0.82 (0.60-1.11)	
Fatal or nonfatal myocardial infarction	21 (0.7)	24 (0.8)	31 (1.0)	38 (1.2)	0.55 (0.32-0.93)	
Fatal or nonfatal stroke	31 (1.0)	39 (1.2)	44 (1.4)	55 (1.7)	0.56 (0.36-0.87)	
Resuscitated cardiac arrest	1 (<0.1)	3 (0.1)	1 (<0.1)	3 (0.1)	0.33 (0.03-3.18)	
Revascularization	27 (0.8)	29 (0.9)	37 (1.2)	45 (1.4)	0.59 (0.37-0.95)	
Heart failure	10 (0.3)	11 (0.3)	11 (0.3)	18 (0.6)	0.55 (0.25-1.19)	
Angina with objective evidence of ischemia	25 (0.8)	31 (1.0)	26 (0.8)	38 (1.2)	0.65 (0.39-1.08)	
Other outcomes	200.000			12.0		
Death from any cause — no. (%)	163 (5.1)	171 (5.4)	179 (5.6)	178 (5.6)	0.91 (0.73-1.12)	
New-onset diabetes — no./total no. (%)	123/2982 (4.1)	109/3001 (3.6)	113/2984 (3.8)	113/2999 (3.8)	1.09 (0.85-1.41)	
Hospitalization no. (%)**						
For cardiovascular causes	141 (4.4)	140 (4.4)	178 (5.6)	191 (6.0)	0.73 (0.59-0.91)	0.005
For noncardior ascular causes	460 (14.0)	418 (13.1)	430 (13.7)	443 (14.0)	1.04 (0.92-1.19)	0.52
First and recurrent events of the second coprimary outcomet	t					
No. of participants with ≥1 event	136	141	176	187		
No. of participants with ≥2 events	29	39	30	59		
No. of participants with ≥3 events	2	4	3	13		
Total no. of events	169	184	211	262	0.66 (0.52-0.84)	0.001



Coprimary and secondary outcomes

Table 2. Primary, Secondary, and Other Outcomes.*						
Outcome	Candesartan- Hydroch lorothiazide plus Rosuvastatin (N = 3180)	Ros uvastatin plus Placebo (N = 3181)	Candesartan- Hydrochlorothiazide plus Placebo (N=3176)	Placebo plus Placebo (N = 31 68)	Candesartan-Hydrochloro Rosuvastatin v s. Placebo j	
					Hazard Ratio (95% CI)	P Value
Coprimary outcomes — no. (%)			NO. 100 (100 (100 (100 (100 (100 (100 (100	West-W	BETTE BETTE BETTE	
First coprimary outcome	113 (3.6)	122 (3.8)†	147 (4.6) ‡	157 (5.0)	0.71 (0.56-0.90)	0.005
Second coprimary outcome	136 (4.3)	141 (4.4)	176 (5.5)¶	187 (5.9)	0.72 (0.57-0.89)	0.003
Secondary outcome — no. (%)	147 (4.6)	159 (5.0)	188 (5.9)	205 (6.5)	0.71 (0.57-0.87)	0.001
Components of the coprimary and secondary outcomes	— no. (%)		200000000000000000000000000000000000000	2-20-11-20-0		
Death from cardi or ascular causes	75 (24)	79 (2.5)	80 (2.5)	91 (2.9)	0.82 (0.60-1.11)	
Fatal or nonfatal my ocardial infarction	21 (0.7)	24 (0.8)	31 (1.0)	38 (1.2)	0.55 (0.32-0.93)	
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Angina with objective evidence of ischemia	25 (0.8)	31 (1.0)	26 (0.8)	38 (1.2)	0.65 (0.39-1.08)	
Other outcomes	21-1-1-1-1		100.400.0	111-11-01-1-0	31373 - 4013 312 - 101	
Death from any cause — no. (%)	163 (5.1)	171 (5.4)	179 (5.6)	178 (5.6)	0.91 (0.73-1.12)	
New-onset di abetes — no./total no. (%)	123/2982 (4.1)	109/3001 (3.6)	113/2984 (3.8)	113/2999 (3.8)	1.09 (0.85-1.41)	
Hospitalization no. (%)**		The second second			11	
For cardiovascular causes	141 (4.4)	140 (4.4)	178 (5.6)	191 (6.0)	0.73 (0.59-0.91)	0.005
For noncardi ov ascular causes	463 (14.6)	418 (13.1)	436 (13.7)	443 (14.0)	1.04 (0.92-1.19)	0.52
First and recurrent events of the second coprimary outco	omett		The state of the s	7,000		
No. of participants with ≥1 event	136	141	176	187		
No. of participants with ≥2 events	29	39	30	59		
No. of participants with ≥3 events	2	4	3	13		
Total no. of events	169	184	211	262	0.66 (0.52-0.84)	0.001

Differences for candesartan/hctz vs dual placebo=NS



Conclusions

- In intermediated risk patients treated for 5.6 yrs, rosuvastatin plus candesartan/hctz reduced cv events vs dual placebo
- Relative RR=29%, absolute RR=1.4%
- NNT to prevent one coprimary outcome event=72, 63 for a secondary outcome event
- Benefit attributed to rosuvastatin although trend towards benefit with BP drugs in the top third of pts with the highest baseline SBP
- ? Diuretic dose too low (should have been chlorthalidone?)
- Polypill may not yet be ready for prime-time

New Engl J Med 2016;374:2009-20,2021-31, and 2032-43



A 62 year old woman with hypertension presents with an office BP of 157/90 and home BPs averaging 148/84. Drugs are HCTZ 50 mg/d, amlodipine 10 mg/d, and losartan 100 mg/d. A previous screen for secondary causes of hypertension was negative. The most effective next add-on drug is:

- A. Bisoprolol 5 mg/d
- **B.** Spironolactone 25 mg/d
- C. Doxazosin 4 mg/d
- D. Minoxidil 2.5 mg/d



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- B. Spironolactone 25 mg/d
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- D. Minoxidil 2.5 mg/d



Resistant Hypertension Definition

- Failure to reach the BP goal (<140/90 mm Hg) based on office BP:
 - Appropriate <u>three-drug</u> regimen that includes a diuretic
 - Full doses of all drugs
 - Adherence to therapy
- Controlled BP requiring ≥ 4 drugs

AHA Professional Education Committee of the Council for High Blood Pressure Research



Resistant Hypertension Prevalence

- Unknown
 - 15 -20% referral clinics and clinical trials
 - ALLHAT
 - 42,418 participants
 - 30% on 3 or more BP medications
 - Control rate 66%

Furberg CD et al. JAMA 2002;288:2981 Loetta G et al. J Hum Hypertens 2008;22:119



Ambulatory blood pressure measurement in resistant hypertension

- 68,045 patients in Spanish registry
 - 8295 (12.2%) with resistant HTN
 - Office BP ≥140/90 mm Hg on 3 drugs with one being a diuretic
 - After ABPM:
 - 5182 (62.5%) True RH
 - 3113 (37.5%) White-Coat RH
 - Cut-point ABPM: 24-hr average BP <130/80 mm Hg

Hypertension 2011;57:898-902



Resistant Hypertension Management Steps

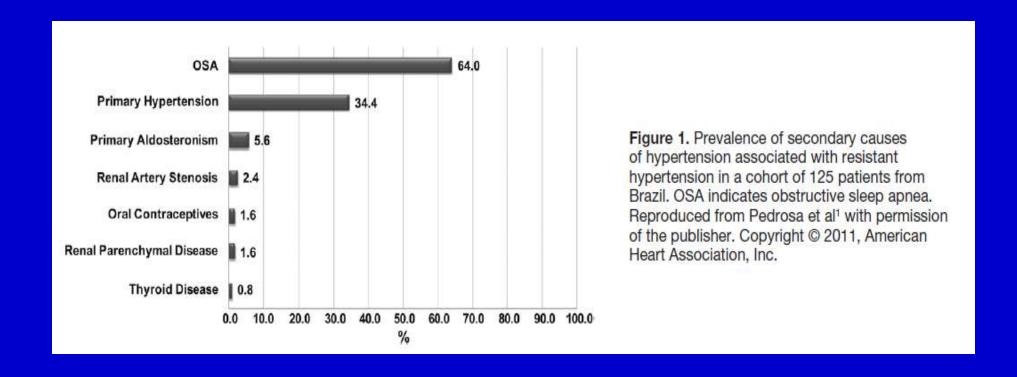
- Exclude white-coat effect, noncompliance, interfering substances and secondary causes
- Control ECV dietary sodium, diuretic appropriate for level of renal function
- Titrate drugs to maximum tolerable doses
- Combine drugs with complimentary mechanisms of action:
 - Diuretic + RAAS blocker + CCB
- Spironolactone as a fourth agent
 - Resulted in significant reduction by office BP or ABPM in metaanalysis of 15 studies (Dahal et al. Am J Hypertens 2015;28:1376-85)



Spironolactone not only lowers blood pressure....



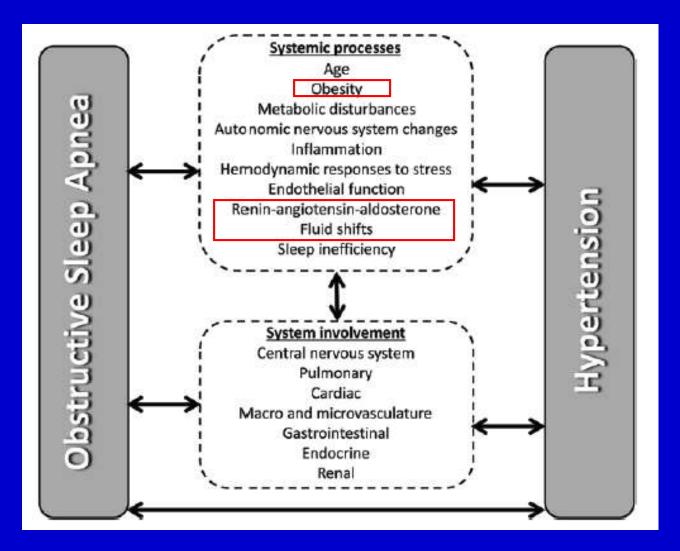
OSA may be the most common secondary cause of hypertension



Konecny, Kara, Somers. Hypertension 2014;63:203-209



Complex interactions between OSA and BP





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www.nature.com/jhh

ORIGINAL ARTICLE

Spironolactone reduces severity of obstructive sleep apnoea in patients with resistant hypertension: a preliminary report

K Gaddam¹, E Pimenta², SJ Thomas³, SS Cofield⁴, S Oparil³, SM Harding⁵ and DA Calhoun³
¹Division of Cardiovascular Diseases, Ochsner Clinic Foundation, New Orleans, LA, USA; ²Endocrine
Hypertension Research Centre and Clinical Centre of Research Excellence in Cardiovascular and Metabolic
Diseases, Princess Alexandra Hospital, University of Queensland School of Medicine, Brisbane, Queensland,
Australia; ³Vascular Biology and Hypertension Program, Division of Cardiovascular Disease, University of
Alabama at Birmingham, Birmingham, AL, USA; ⁴Department of Biostatistics, University of Alabama at
Birmingham, Birmingham, AL, USA and ⁵Division of Pulmonary, Allergy and Critical Care Medicine and
Sleep/Wake Disorders Center, University of Alabama at Birmingham, Birmingham, AL, USA



Table 2 Characteristics before and after spironolactone treatment				
Characteristics	Baseline	8 weeks	P-value	
Weight, lbs	243.0 ± 32.4	239.9 ± 29.4	0.03	
	42.1 ± 3.5	41.2 + 3.4	0.195	
Clinic SBP, mm Hg	145 ± 18	124 ± 16	<0.001	
Clinic DBP, mm Hg	81 ± 16	72 ± 9	0.04	
Ambulatory daytime SBP, mm Hg ^a	150 ± 14 85 ± 15 142 ± 16	134 ± 18	0.04	
Ambulatory daytime DBP, mm Hg ^a		75 ± 11	0.06	
Ambulatory night time SBP, mm Hg ^a		120 ± 23	0.02	
Ambulatory night time DBP, mm Hg ^a	77 ± 12 147 ± 13	64 ± 13	0.016	
24-h systolic blood pressure, mm Hg ^a		130 ± 19	0.025	
24-h diastolic blood pressure, mm Hg ^a No. of antihypertensive medications Serum creatinine	82 ± 14 4.3 ± 1.1 1.2 ± 0.3	72 ± 11 4.5 ± 1.0 1.3 ± 0.2	0.051 0.76 0.035	
Serum potassium, mEq l ⁻¹	4.0 ± 0.3	4.4 ± 0.5 14.3 ± 13.8	0.05	
Plasma renin activity, ng ml ⁻¹ h ⁻¹	1.4 ± 2.1		0.005	
BNP, pgml ⁻¹ AHI, events h ⁻¹	17.3 ± 12.7	12.6 ± 18.8	0.24	
	39.8 ± 19.5	22.0 ± 6.8	<0.001	
Hypoxic index, %	13.6 ± 10.8	6.7 ± 6.6	0.04	
Supine AHI, events h ⁻¹	63.2 ± 28.7	40.8 ± 19.3	0.007	
REM AHI, events h ^{-1a}	55.7 ± 27.9	33.9 ± 19.7	0.003	

Gaddam et al. J Hum Hypertens 2010;24:532-537



Changes in OSA parameters after treatment with spironolactone

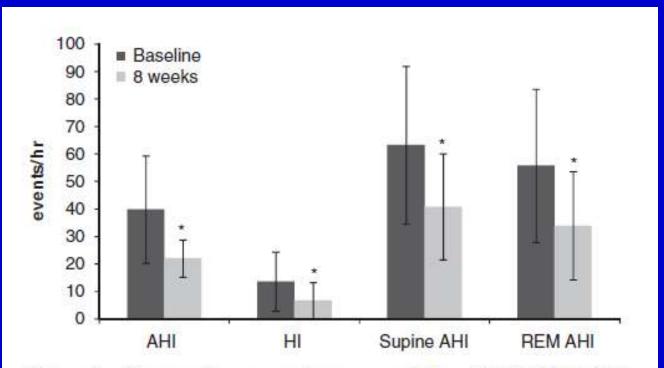


Figure 1 Changes in apnoea—hypopnoea index (AHI) (39.8 \pm 19.5 vs 22.0 \pm 6.8); hypoxic index (HI) (13.6 \pm 10.8 vs 6.7 \pm 6.6); supine AHI (63.2 \pm 28.7 vs 40.8 \pm 19.3); and rapid eye movement sleep (REM) AHI (55.7 \pm 27.9 vs 33.9 \pm 19.7) at 8 weeks (light grey bars) compared with baseline (dark grey bars). Values, mean \pm s.d. *Different compared with baseline, P<0.05.

How could spironolactone improve OSA indices?

- OSA is associated with significant extracellular volume expansion-typically not controlled on a thiazide diuretic
- At night when supine, fluid mobilization leads to peripharyngeal fluid accumulation resulting in increased pharyngeal obstruction-aldosterone must play a role
- Other evidence of a role for volume overload:-volume reduction improves OSA in:
 - CHF
 - ESRD pts switched to nocturnal hemodialysis or peritoneal dialysis



Spironolactone versus placebo, bisoprolol, and doxazosin to determine the optimal treatment for drug-resistant hypertension (PATHWAY-2): a randomised, double-blind, crossover trial

Bryan Williams, Thomas M MacDonald, Steve Morant, David J Webb, Peter Sever, Gordon McInnes, Ian Ford, J Kennedy Cruickshank, Mark J Caulfield, Jackie Salsbury, Isla Mackenzie, Sandosh Padmanabhan, Morris J Brown, for The British Hypertension Society's PATHWAY Studies Group*

Hypothesis: resistant hypertension is predominantly caused by sodium retention, therefore, spironolactone will be the most effective add-on agent and its effectiveness will be inversely proportional to the baseline plasma renin level



Study Design

- Inclusion criteria
 - Clinic SBP ≥ 140 (≥ 135 for diabetics) and home SBP ≥ 130 (mean of 18 readings over 4 days)
 - Maximally tolerated doses of ACEI/ARB + CCB + diuretic
 - Ages 18-79 yrs
- 4 week placebo run in, then randomized to spironolactone (25-50 mg), doxazosin (4-8 mg), bisoprolol (5-10 mg) or placebo
- All subjects receive all drug in randomized fashion for 12 weeks (first 6 at low dose, second 6 at higher dose)
- Primary outcome measurement: average home SBP measured in triplicate AM and PM for 4 consecutive days prior to a study visit (Micolife® automated device)



Lancet 2015;386:2059-68

Baseline Characteristics

	Mean (SD) or N (%)
Age (years)	61-4 (9-6)
Sex .	
Male	230 (69%)
Female	105 (31%)
Weight (kg)	93-5 (18-1)
Smoker	26 (7-8%)
Home	
Systolic blood pressure (mm Hg)	147-6 (13-2)
Diastolic blood pressure (mm Hg)	84-2 (10-9)
Heart rate (beats per min)	73-3 (9-9)
Clinic	
Systolic blood pressure (mm Hg)	157-0 (14-3)
Diastolic blood pressure (mm Hg)	90-0 (1-5)
Heart rate (beats per min)	77-2 (12-2)
24 h urine (mmoV24 h)	
Sodium	137-1 (71-8)
Potassium	70-5 (29-5)
Blood electrolytes (mmol/L)	
Sodium	139-6 (3-0)
Potassium	4-1 (0-5)
eGFR (mL/min)	91-1 (26-8)
Diabetic	46 (14%)
FR=estimated glomerular filtration rate.	

PATHWAY-2 study (n=335)



Results: blood pressure

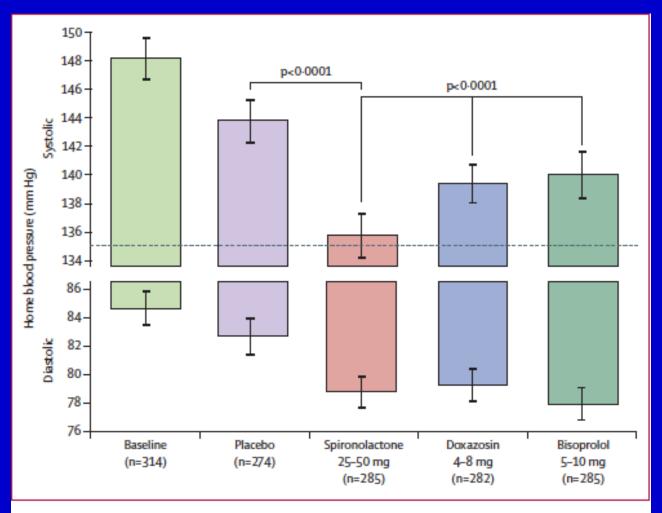


Figure 2: Home systolic and diastolic blood pressures comparing spironolactone with each of the other cycles



Results: blood pressure

	Blood pressure (mm Hg)	Change from baseline (mm Hg)
Mean		
Spironolactone	133-5 (132-3 to 134-8)	-14-4 (-15-6 to-13-1)
Doxazosin	138-8 (137-6 to 140-1)	-9·1 (-10·3 to -7·8)
Bisoprolol	139-5 (138-2 to 140-8)	-8-4 (-9-7 to -7-1)
Placebo	143-7 (142-5 to 145-0)	-4·2 (-5·4 to -2·9)
Mean differences		
Spironolactone vs placebo	-10·2 (-11·7 to -8·74)	p<0.0001
Spironolactone vs mean bisoprolol and doxazosin	-5·64 (-6·91to-4·36)	p<0.0001
Spironactone vs doxazosin	-5·30 (-6·77 to-3·83)	p<0.0001
Spironolactone vs bisoprolol	-5·98 (-7·45 to -4·51)	p<0.0001
		22

Data are mean (95% CI). Sensitivity analysis using only the mean home systolic blood pressure at the final visit of each cycle (week 12).

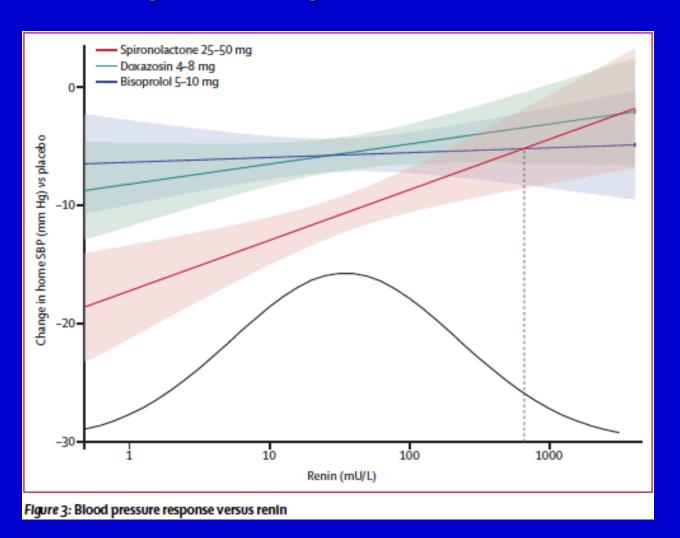
Table 3: Home systolic blood pressure at final visit of each cycle

	Blood pressure (mm Hg)	p value
Spironolactone	-3-86 (-5-28 to -2-45)	<0.0001
Doxazosin	-0.88 (-2.32 to 0.56)	0-23
Bisoprolol	-1·49 (-2·94 to-0·04)	0-04
Placebo	-0.68 (-2.10 to 0.75)	0-35

Lancet 2015;386:2059-68



The lower the baseline renin, the better the response to spironolactone





Results: Adverse Events

	Spironolactone	Doxazosin	Bisoprolol	Placebo	p value*
Serious adverse events	7 (2%)	5 (2%)	8 (3%)	5 (2%)	0.82
Any adverse event	58 (19%)	67 (23%)	68 (23%)	42 (15%)	0.036
Withdrawals for adverse events	4 (1%)	9 (3%)	4 (1%)	3 (1%)	0.28
Date of the state					

Data are n (%). *p values for Fisher's exact test. The most common adverse events in at least 5% of patients on any treatment are shown in appendix p 12.

Table 5: Adverse events and withdrawals

Discontinuations due to renal impairment, hyperkalemia, and gynecomastia were not increased with spironolactone vs all others

- Mean change in serum K was 0.45 mEq/L
- 6 (2%) of the 285 patients exposed to spironolactone developed a serum potassium on a single occasion of > 6 mEq/L (maximum 6.5)



Conclusions

- Spironolactone was more effective than doxazosin or bisoprolol in reducing home blood pressure in patients with resistant hypertension
- Spironolactone was most effective in patients with the lowest baseline plasma renin (supporting the hypothesis of volume expansion in patients with resistant hypertension)
- Despite adding spironolactone to a regimen of an ACEI or ARB, the risk of hyperkalemia was low (though should be followed closely, at least initially)
- It is my go-to drug, particularly in the setting of OSA and obesity



Allopurinol has been demonstrated to do all of the following except:

- A. Lower blood pressure
- B. Reduce the development of new kidney disease and delay the progression of chronic kidney disease
- C. Reduce the risk of coronary events and stroke
- **D.** Increase the risk of Type 2 Diabetes Mellitus



Allopurinol has been demonstrated to do all of the following except:

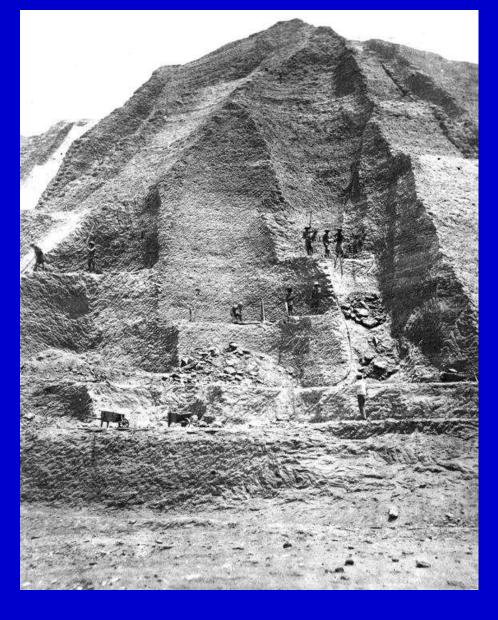
- A. Lower blood pressure
- B. Reduce the development of new kidney disease and delay the progression of chronic kidney disease
- C. Reduce the risk of coronary events and stroke
- D. Increase the risk of Type 2 Diabetes Mellitus



URIC ACID METABOLISM

- product of purine metabolism
- in most mammals, degraded by hepatic enzyme, urate oxidase (uricase) to more soluble allantoin which is freely excreted in urine
- Excreted as dry mass in feces (guano) of birds and reptiles to conserve water
- humans and great apes: mutations occurred >5 million years ago rendering uricase gene nonfunctional; result: higher UA levels than other mammals (< 2 mg/dl in latter)





Workers in the 1860s excavate a "mountain" of guano more than 60 feet tall. Photograph © Smithsonian Institution



Serum uric acid is a predictor of:

- Hypertension in pre-teens and adolescents
- CV events:
 - in the general population
 - in patients with hypertension
 - In patients with pre-existing CVD

Hypertension 2005;45:34-38.

Hypertension 2003;41:1183-1190

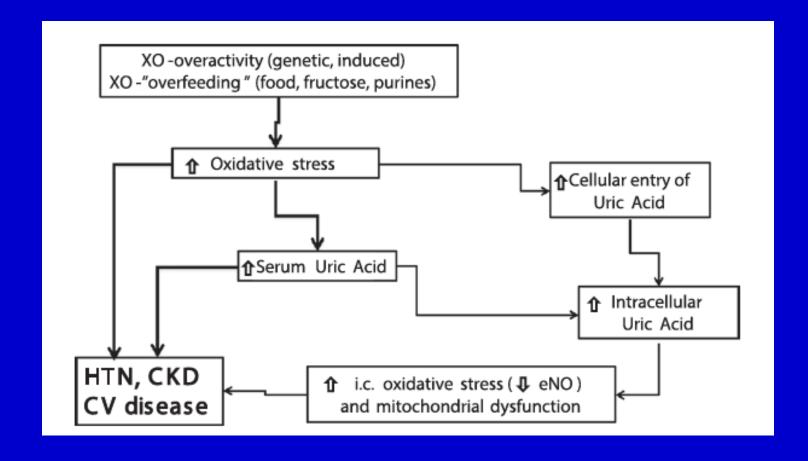
Hypertension 2006;48:1031-1036.

Hypertension 2006;48:1037-1042.

J Hypertens 2007;25:1583-1589.



Uric acid is a player in cardiovascular and kidney disease



Hypertension 2016;67:496-498

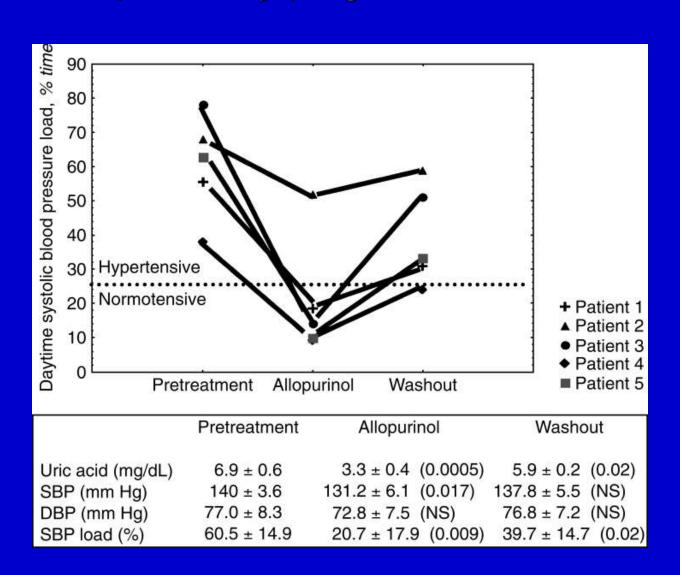


Clinical studies of allopurinol

- Lowers blood pressure in adolescents (JAMA 2008;300:924) and older adults (J Clin Hypertens 2013;15:435)
- Delays progression of renal disease and reduces cardiovascular risk and hospitalizations in patients with CKD (Clin J Am Soc Nephrol 2010;5:1388)
- Decreases the new onset of renal failure in people 65 years or older (the higher the dose and the longer the treatment the better)(Ann Rheum Dis 2016;0:1)



Treatment of newly diagnosed hypertensive adolescents with allopurinol, 200mg/d, for one month: a pilot study (Feig et al: KI 2004;66:281-287)





Allopurinol and Cardiovascular Outcomes in Adults With Hypertension

Rachael L. MacIsaac, Janek Salatzki, Peter Higgins, Matthew R. Walters, Sandosh Padmanabhan, Anna F. Dominiczak, Rhian M. Touyz, Jesse Dawson

Hypertension 2016;67:535-540



Methods

- United Kingdom Clinical Research Practice Datalink (UK CRPD):
 - Demographics, diagnoses, medical histories, prescriptions from 500 primary care practices (n > 3.4 million patients)
- Identified 2400 pts 65 yrs or older and on allopurinol (presumably for gout) and 42,000 matched controls not on allopurinol. All subjects had a dx of hypertension
- Exclusions: CKD
- Primary outcomes (new event over 10 yrs follow-up):
 - Stroke
 - Cardiac event (MI or acute coronary syndrome



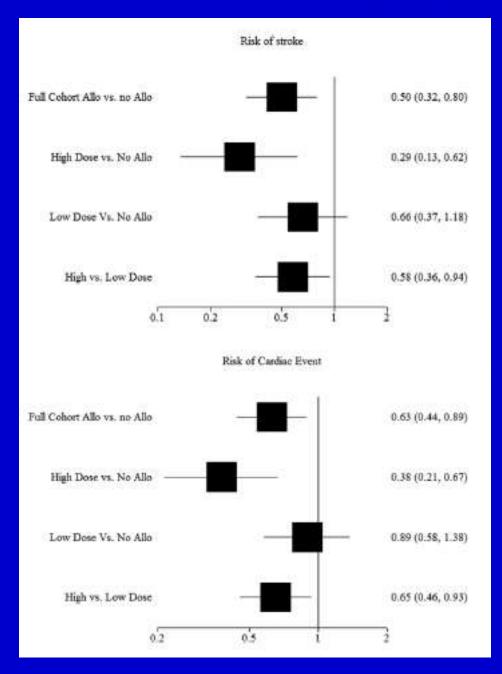
Baseline characteristics

		Any Allopurinol Exposure
	<u>No</u>	<u>Yes</u>
• Age (y)	73	73
• BP	156/86	157/86
• Sex (% female)	38	38
• Gout	?	?

Yes group: higher BMI, higher creatinine, less CCB use, more DM and ACEI use, same amount of diuretic use



Results



Allopurinol dosing: Low: <300 mg/d High: 300-600 mg/d

Serum uric acid levels:

Pre-Rx only available in onethird of allopurinol group with f/u levels available in one-third of that group

Mean 8.6→6.2 mg/dl

Hypertension 2016;67:535-540

Conclusions

- Allopurinol was associated with a reduced risk of both stroke (by 50%) and cardiac events (by 39%) in patients 65 y or older with hypertension
- This benefit appears limited to patients on allopurinol doses of 300 mg/d or higher
- Limitations
 - retrospective
 - Indications for allopurinol not specified
 - Gout is an independently-associated risk factor for CVD
 - Limited serum uric acid data
- Need for prospective clinical trials



Take-home Points

- In hypertensive patients age 75 and older, decreasing systolic blood pressure towards 120 mm Hg may be well tolerated and beneficial (CVD, all-cause mortality)
- In patients with intermediate cv risk factors and without clinical cvd, treatment with rosuvastatin (regardless of LDL-C) may be beneficial
- Spironolactone is the best add-on drug in patients with resistant hypertension
- Consider the use of allopurinol in older hypertensive patients with hyperuricemia, especially in the setting of chronic kidney disease







On the other hand, consider the consequences of the unrestricted harvesting of trees...





Thank you

vcanzanello@mayo.edu



Stress Management Training and Cardiovascular Health: Have we found the Holy Grail?

Jon C. Ulven, PhD, LP Chair of Adult Psychology Sanford Health Fargo

- I work for a not-for-profit health system (Sanford Health System)
- I have no competing interests to disclose

Disclosures

- Licensed Psychologist with degree from KU
- Practicing in Internal Medicine since 2008
- Clinical Skills Development Team Member for a \$12 million innovation award from the Center for Medicare and Medicaid Services focused on improving primary care services (2012 to 2015) at Sanford Health
- Chronic Care Professional Certificate (2014) from Health Sciences Institute
- Certificate in Primary Care Behavioral Health (2011) from University of Massachusetts Medical Center

Why am I talking to you about stress management?

- What is the research-based link between stress management training and reduction of adverse cardiovascular events?
- Describe stress management training, identifying relevant components
- Practice relaxation exercises
- Illustrate ways to increase patient readiness to engage in stress management training

Learning Objectives

 Learning to monitor and control muscular tension developed by Dr. Edmund Jacobson in the 1920s

https://www.anxietybc.com/sites/default/files/MuscleRelaxation.pdf

Progressive Muscle Relaxation

- CVD includes CAD, cardiomyopathy, valvular heart disease, pericardial disease, arteriosclerosis, atherosclerosis, aneurism, high blood pressure, stroke, and peripheral artery disease
- Remains the #1 case of death in the US
- # of deaths attributable to CVD has dropped 30% from 2001 to 2011 but still accounts for 1 in 3 deaths
- More than 1/3 of us have some form of CVD

Mozaffarian et al., 2015

Cardiovascular Disease (CVD)



- We can't control age, sex, race, and heritable risk, but we can decrease the risk of developing CVD by targeting...
 - Physical factors
 - Behavioral factors
 - Emotional and Cognitive factors
 - Environmental Factors

Biopsychosocial Factors in CVD

- Blood Pressure
 - Essential hypertension is #1 primary care diagnosis (CDC, 2010)
- Cholesterol
 - "Lifestyle changes" are foundational
 - Statin therapy for patients who can tolerate them

Physical Factors

Estimated Mortality Reductions for people with CVD (Iestra, et al., 2005)

- Decreased Tobacco Use (35%)
- Reduced Overeating; Improved Dietary Changes (45%)
- Increased Physical Activity (25%)
- Moderate Alcohol Consumption (20%)
- Medication Adherence (similar to above)

Behavioral Factors

Depression

- Tiredness, lack of energy, and irritability are often mistaken for effects of CVD
- Important to screen for depressive symptoms
- Tx of Depression has not been shown to reduce risk of future cardiac events yet, so goal is for improved quality of life

Anxiety

- Chronic problem in 20-25% of patients with CAD
- Associated with increased risk of sudden cardiac death

Type A

- As classically defined is NOT related to CAD
- Hostility (i.e., anger, cynicism, mistrust) IS

Emotional and Cognitive Factors

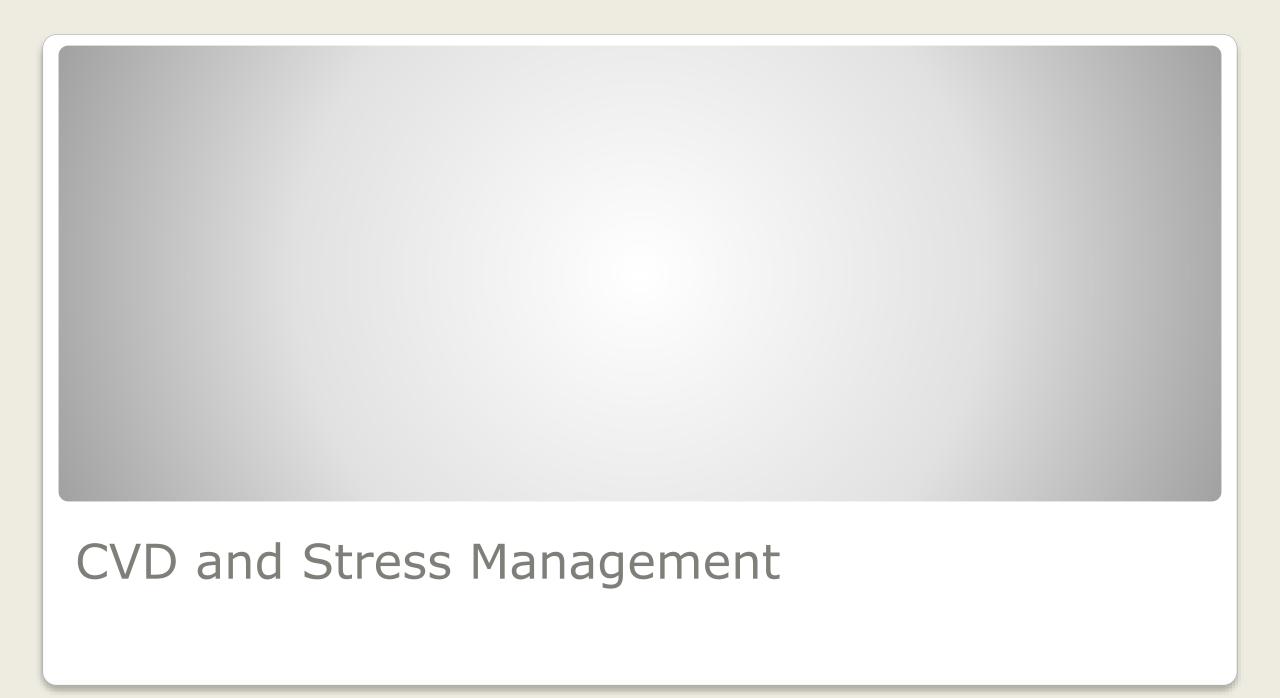
- Risk Factors
 - Small social support networks
 - Low levels of perceived support
 - Social isolation
 - Low socioeconomic status
- Some evidence that improving social support improves many problems (e.g., cancer, weight loss, recovery from surgery, birth preparation)
 - Often methodological flaws in this research (Hogan et al., 2002)
- Quality and Quantity of Social Relationships is as Beneficial as Smoking Status (Holt-Lunstad, Smith, and Layton, 2010)

Environmental Factors

 Mind-body intervention that uses imagination/memory to simulate sensory experiences

 https://psychcentral.com/lib/imagery-basic-relaxationscript/

Guided Imagery for Stress Management



• Elevated levels of stress are associated with greater risk of death and non-fatal events (Rozanski et al., 2005; Rosengren et al., 2004)

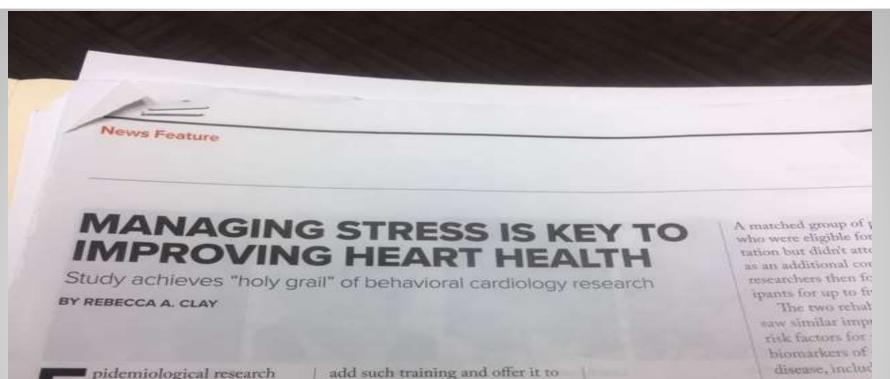
CVD and Stress

- Women with CVD who participated in a stress management intervention had a 3 times greater survival rate after 7 years compared to usual care (Orth-Gomer et al., 2009)
- Cochrane Systematic Review (2014) found that psychological interventions were associated with fewer deaths due to cardiac causes compared to usual care

CVD and Stress Management

- Recent meta-analysis suggested that CR reduces cardiovascular mortality, hospitalizations, and improves quality of life (Anderson et al., 2016)
- Core components of CR: exercise, medical management of BP and lipids, nutritional counseling, and smoking cessation
- Stress management training is not routinely part of CR due to...

Cardiac Rehabilitation (CR)



pidemiological research has long shown a link between stress and heart disease. What has been missing is strong evidence showing that reducing stress actually

all participants, he says. "Stress management isn't just for those who are distressed, but has benefits for everybody," he says. In the study, known as

inflammation variability. B lower rates events that The stress

"Holy Grail" of Behavioral Cardiology Research

- RCT 151 outpatients with Coronary Heart Disease randomized to 12 weeks of CR or CR plus Stress Management Training (SMT) and followed for over 3 years
- CR + SMT had better medical outcomes and had greater reductions in stress than CR alone
- Conclusion SMT should be incorporated into CR
- http://circ.ahajournals.org/content/early/201 6/03/10/CIRCULATIONAHA.115.018926

Blumenthal et al. (2016)

- Goal of SMT is to reduce demands and increase coping ability
- Combination of Education, Group Support, and CBT (brief lectures, role playing, weekly homework, group discussion)
- Reducing demands: prioritizing, time management, establishing personal values, avoiding stress-producing situations
- Modifying responses: progressive muscle relaxation, visual imagery, reducing distorted thinking, assertive communication, problem-solving

Stress Management Training (SMT)

 Mindfulness is a non-religious skill of being aware of the present moment in a non-judgmental way

https://www.reading.ac.uk/web/files/counselling/Mindfulness 2015.pdf

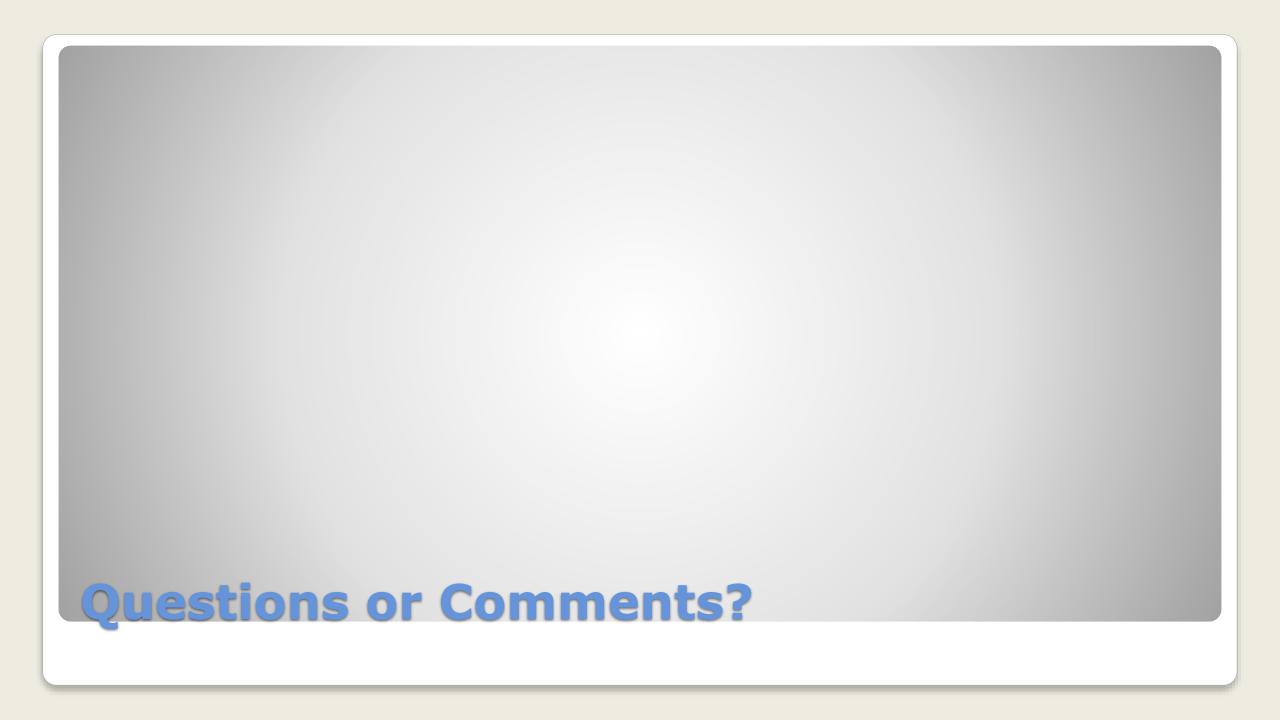
3 minute Breathing Space

- Be a good Guide to your patients
- Invite your patients to work together to find ways to reduce CVD risk
- Assess the biopsychosocial factors impacting your patients
- Equip yourself with knowledge to discuss with patients what works in reducing CVD risk
- Assess their readiness to change and consider focusing on one area at a time rather than trying to change multiple behaviors at once (Ebrahim et al., 2011)

Increasing Engagement

- There are numerous barriers to health, but there are also numerous ways to meaningfully intervene – consider the biopsychosocial factors
- Stress management improves clinical outcomes when added to Cardiac Rehab
- Patients often have misconceptions about stress management and psychological interventions, especially regarding improving overall health. What are your thoughts?

Summary Points



Thank you for attending the 2017 Hypertension Summit – Advanced Practice Session!!

Please complete: Evaluation Form

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NORTH DAKOTA

DEPARTMENT of HEALTH

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